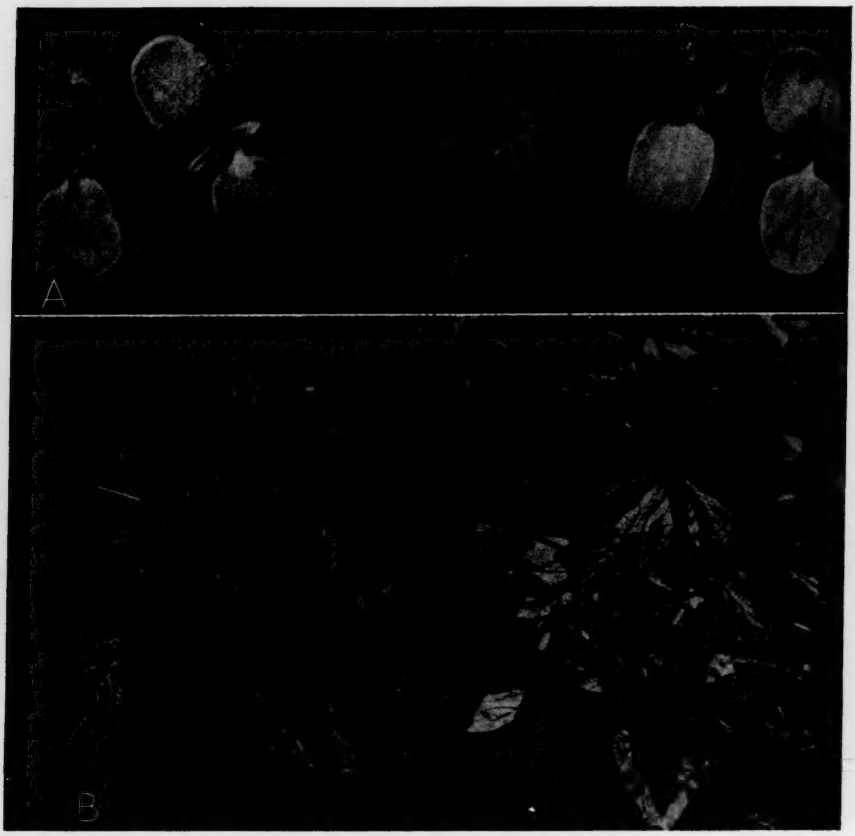
The Journal of HEREDITY



"OPEN SESAME"—"CLOSE SESAME"

(SEE PAGE 149)

Hereditary Hip Disease Mutation Mink Genetics

VOLUME 37 MAY, 1946 NUMBER 5

The American Genetic Association

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The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and 1507 M St., N. W., Washington, D. C. Address all communications regarding editorial matters to 1507 M. St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1507 M St., N. W., Washington 5, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 29, 1918.

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The
Journal of Heredity

Vol. XXXVII MAY, 1946 No. 5

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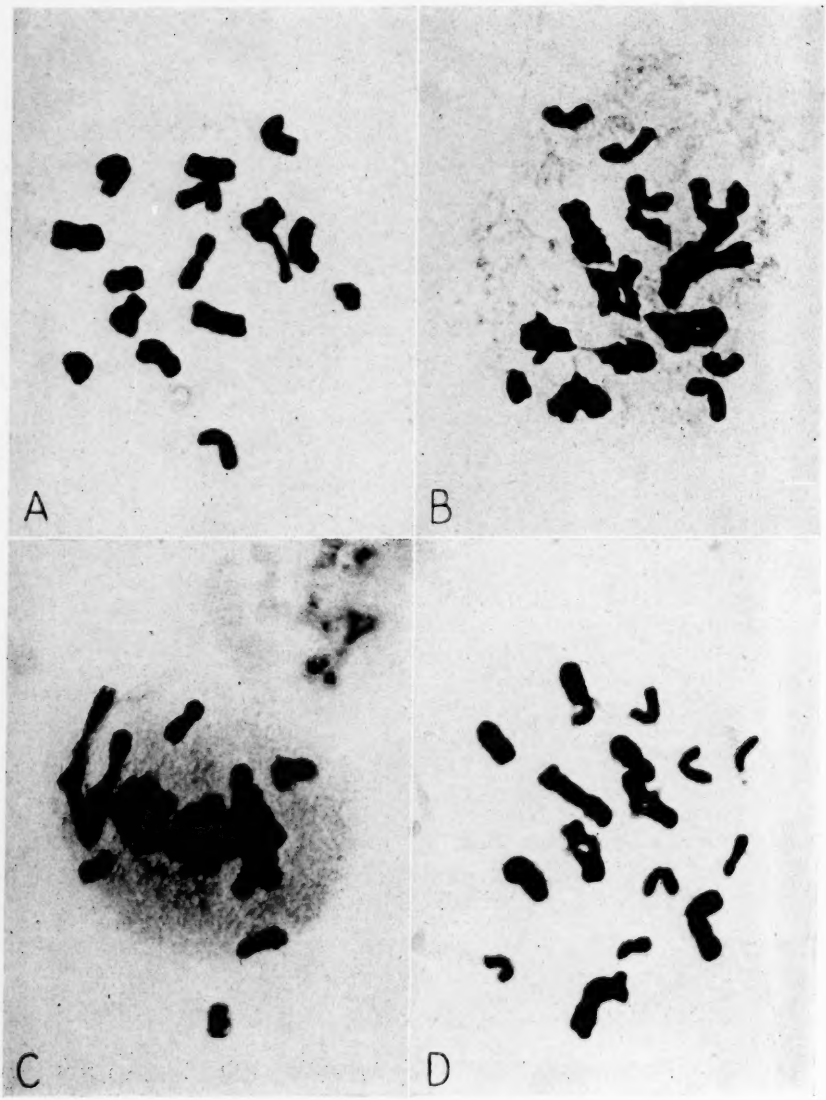

TRIPLOID POLLEN MOTHER CELLS

Figure 1

Chromosome plates in pollen mother cells of triploid species hybrids of onions. *A*—Amphidiploid × *Allium fistulosum,* showing eight bivalents and eight univalents. *B*—Amphidiploid × *A. cepa,* showing eight bivalents and eight univalents. *C*—Amphidiploid × *A. cepa,* showing a trivalent. *D*—Amphidiploid × *A. cepa* plant with 25 chromosomes, showing nine bivalents and seven univalents. The difference in appearance of the chromosome pairs shown in *A* and *B* is due to the type of pairing characteristic of the reciprocal hybrid in this cross.

A CYTOLOGICAL STUDY OF SOME TRIPLOID ONION PLANTS

ALFRED E. CLARKE AND HAZEL H. MCKAY*

IN 1942 Jones and Clarke[6] described an amphidiploid plant that arose from the cross *Allium cepa* L. var. Australian Brown × *A. fistulosum* L. type Nebuka. Walker, Jones, and Clarke[16] found that this amphidiploid is highly resistant to onion smut (*Urocystis cepulae* Frost) and also (unpublished data) to onion pink root (*Phoma terrestris* Hansen). Consequently, it is being used as a possible means of incorporating smut resistance and pink root resistance into stocks of commercial value. Backcrosses of the amphidiploid to both parent species were made in the course of this breeding program and gave rise to the triploid plants described in this paper.

Allium cepa and *A. fistulosum* differ in the type of chromosome pairing that they exhibit, because of a marked difference in chiasma behavior between the two species. In *A. cepa* the chiasmata are randomized, and at diakinesis and first metaphase the chromosome pairs are held together by terminal or subterminal chiasmata. Levan[8] and later Emsweller and Jones[2] showed that in *A. fistulosum* there is a random distribution of chiasmata in the earlier prophase stages, but later the chiasmata become localized at the kinetochore. Consequently, the bivalents are ring- or rod-shaped in *A. cepa*, whereas they are typically cruciform in *A. fistulosum*.

First-generation populations of hybrids between *A. cepa* and *A. fistulosum* were studied by Levan,[10] Emsweller Jones,[2] and Maeda.[14] These authors noted marked cytological differences in the behavior of their respective groups of hybrids, but in all cases at first metaphase in the first generation the random type of chiasmata was found, as in the *A. cepa* parent. Cochran[1] has studied hybrids between *A. ascalonicum* L. and *A. fistulosum*. *A. ascalonicum* is closely related to *A. cepa* and is considered by many botanists to be a subspecific or varietal form of the latter. It has the random type of chiasmata, and according to Cochran the first-generation plants also have the random type.

When the first-generation plants resulting from *A. cepa* × *A. fistulosum* are backcrossed to *A. cepa* all the progeny have random chiasmata. When backcrossed to *A. fistulosum* some plants have all random chiasmata, others all localized, and still others both random and localized in the same cell. Maeda[14] obtained both second-generation and backcross plants that showed randomized and localized pairs in the same cell, the number of localized pairs being constant in any given plant. Emsweller and Jones[3] found that their plants were predominantly of one type or the other, giving a ratio of 1 random : 1 local. They suggested first that the type of chiasma formation was governed by a gene, but their later work indicated that this apparent 1 : 1 ratio resulted from the elimination of practically all gametes other than those with chromosomes predominantly *A. cepa* or *A. fistulosum*.

Colchicine-induced autotetraploids of both *A. cepa* and *A. fistulosum* were obtained by Toole and Clarke.[15] Quadrivalents, trivalents, and univalents were more frequent in *A. cepa* than in *A. fistulosum*: these species averaged 6.5 and 2.5 polyvalents per cell, respectively. In a natural amphidiploid hybrid between these two species, described by Jones and Clarke,[6] multivalent associations were not found at first meiotic metaphase, and the pairing of the bivalents was regular. Some pairs with ran-

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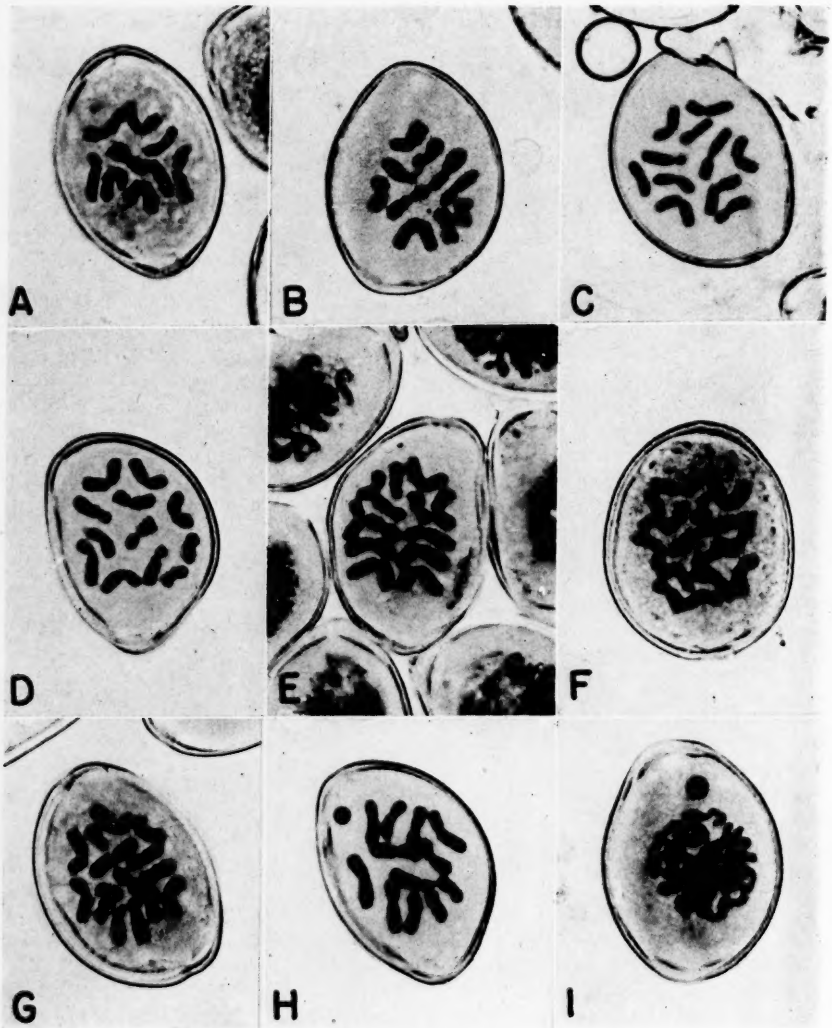

CHROMOSOMES OF TRIPLOIDS

Figure 2

Microspore division figures in triploid species hybrids of onion. *A* to *G*, Chromosome counts of 9, 10, 11, 12, 13, 14, and 15, respectively. *H*, Twelve chromosomes plus a chromosome fragment. *I*, Micronucleus formed possibly by a chromosome or a chromosome fragment

domized and others with localized chiasmata were observed in the same cell. In the tetraploid described by Levan[13] polyvalent associations occurred, but they averaged less than one per cell, and only randomized chiasmata were found. As pointed out by Emsweller and Jones[4] this difference suggests that the tetraploid studied by Jones and Clarke was a true amphidiploid arising from the doubling of the chromosome number of a first-generation zygote, whereas that of Levan probably arose from the union of two unreduced gametes or the plant was derived from spontaneous chromosome doubling in a zygote formed by the union of two gametes composed predominantly of *A. cepa* chromosomes.

Levan[12] found that triploid forms of *A. amplectens* Torr. are characterized by an almost absolute lack of chiasmata at meiosis, a condition he attributed to genetic causes and not to failure of pairing, because the pachytene chromosomes are paired in normal fashion for triploid species.

Levan[9,11] obtained a triploid *A. schoenoprasum* L. by crossing an autotetraploid form with a diploid. Crosses between these artificial triploids and the diploid gave different results, according to whether the diploid or the triploid was used as the female parent. When the triploid was used as the mother plant, all chromosome numbers between 16 and 24 were obtained in the progeny, with the exception of the 20-chromosome type. In the reciprocal cross, when the diploid functioned as the female parent, most of the progeny were diploids.

In a small second-generation progeny from the hybrid *A. cepa* × *A. fistulosum* Levan[13] obtained two triploid plants. In a detailed study of meiosis in one of these triploids, trivalents were found to occur with great frequency, but bivalents and univalents were also observed.

Materials and Methods

The amphidiploid *A. cepa* × *A. fistulosum* was backcrossed both to *A. cepa* and to *A. fistulosum*, the amphidiploid being used as the female parent in all crosses. These crosses were made in the greenhouse of the Plant Industry Station, Beltsville, Md.; flies were used as pollinating agents, as described by Jones and Emsweller.[7]

Preparations of pollen mother cells for microscopic study were made by the usual propiono-carmine smear technic, using a one per cent solution of Clarase, a proprietary enzyme complex, to obtain a better spread of the chromosomes, as described by Emsweller and Stuart.[5] Later a one percent solution of Pectinol was substituted for the Clarase.

Experimental Results

The somatic number of chromosomes is 16 in both *A. fistulosum* and *A. cepa* and 32 in the amphidiploid plant. The gametes formed by the amphidiploid contain 16 chromosomes, eight being derived from the *A. fistulosum* parent and eight from *A. cepa*. Consequently, 16 of the 24 chromosomes in the triploid resulting from amphidiploid × *A. fistulosum* are derived from *A. fistulosum* and the remaining eight from *A. cepa*, whereas in the triploid resulting from amphidiploid × *A. cepa*, eight are derived from *A. fistulosum* and 16 from *A. cepa*.

At first metaphase the triploid plants regularly form eight bivalents and eight univalents. The bivalents line up in regular fashion at the equator of the spindle, but the univalents remain scattered. The plants of the backcross amphidiploid × *A. fistulosum* for the most part produce bivalents that are intermediate in type of pairing. The two members of each pair lie closely associated throughout their entire length and are probably held together by both terminal or subterminal chiasmata and by chiasmata at the kinetochore region (Figure 1*A*). The plants of the backcross amphidiploid × *A. cepa* produce some pairs with side-by-side pairing, but like *A. cepa* other pairs have random chiasmata only (Figure 1*B*). Though there is a great deal of variation in the pairing behavior of the bivalents between different figures, even on the same slide, nevertheless the difference in the appearance of the bivalents in the back-

crosses of *A. cepa* and *A. fistulosum* is sufficiently marked so that no difficulty was experienced in distinguishing between them.

Presumably, at first metaphase in the plants resulting from the amphidiploid × *A. fistulosum* backcross, the eight chromosomes of *A. fistulosum* derived from the amphidiploid female parent are paired with the eight chromosomes of *A. fistulosum* derived from the diploid male parent, whereas the eight chromosomes of *A. cepa* derived from the amphidiploid are left unpaired. Similarly, in the backcross between amphidiploid × *A. cepa* the bivalents consist of the *A. cepa* chromosomes and the univalents of those of *A. fistulosum*. This interpretation is at least partially confirmed by the behavior of the satellite chromosomes. In both species the kinetochore of the satellite chromosome is submedian in position, dividing the chromosome into a long and short arm, whereas in the remaining chromosomes the arms are more nearly equal in length. In both species the satellite is carried on the short arm of the chromosome. However, it is possible to distinguish the satellite chromosomes of *A. fistulosum* from that of *A. cepa*, because in *A. fistulosum* the short arm of the chromosome is even shorter in proportion to the long arm than in *A. cepa*. Also, at first meiotic metaphase the satellite of the chromosome of *A. fistulosum* is much more conspicuous than that of *A. cepa*. Examination of a large number of figures has shown that in the backcross of amphidiploid × *A. fistulosum* the satellite chromosome of *A. cepa* is present as a univalent, whereas in the backcross of amphidiploid × *A. cepa* the satellite chromosome of *A. fistulosum* is a univalent.

Eight bivalents and eight univalents were found in almost all figures that could be counted but an occasional trivalent was observed (Figure 1*C*). Trivalents were, however, very uncommon in this material.

One plant of amphidiploid × *A. cepa* was found with a root tip count of 25 chromosomes instead of the expected 24. Probably the simplest explanation to account for the origin of this plant is to assume that a 17-chromosome gamete from the amphidiploid parent united with a normal eight-chromosome gamete from the pollen parent *A. cepa*. At first meiotic metaphase nine bivalents and seven univalents are formed (Figure 1 *D*). Because there are only seven instead of the theoretically expected eight univalents of *A. fistulosum* it seems highly probable that the extra chromosome was derived originally from *A. fistulosum*, so that the extra bivalent is made up of a pair of chromosomes of *A. fistulosum*.

The formation of bridges and fragments during meiosis is of frequent occurrence in these triploids. At first anaphase in the backcross amphidiploid × *A. fistulosum*, 42 bridges were found in a count of 2,161 figures (1.94 per cent); in the backcross amphidiploid × *A. cepa*, 85 bridges were found in 2,820 figures (3.01 per cent). The greater number of bridges found in triploid amphidiploid × *A. cepa* may be considered significant, because the chi-square goodness-of-fit test gave a *P* value of between 0.05 and 0.01, indicating that a fit as poor or poorer would be expected on the basis of chance alone in less than one out of every 20 trials. This suggests that there is more chromosome irregularity in the backcross with *A. cepa* than with *A. fistulosum*, but further work would be necessary to determine if this is invariably true.

Chromosome counts in the first microspore division of the triploids varied from eight to 16 (Figure 2*A-G*). With random assortment of the univalents the distribution of the chromosomes should approximate the binominal expansion $(a + b)^8$, where a represents the number of univalents going to one pole and b the number going to the other. The results actually obtained are shown in Table I. Some microspores that possessed a small rounded body, presumably a chromosome fragment, in addition to the regular chromosome complement (Figure 2*H*), have been omitted from the table. The plants of amphi-

diploid × *A. fistulosum* gave a satisfactory fit to the theoretically expected numbers, the *P* value being between 0.70 and 0.50. However, the plants of amphidiploid × *A. cepa* gave a poor fit, the *P* value being less than 0.01, because of the more frequent occurrence of the lower chromosome numbers than would be expected on the basis of random assortment. The two populations probably differ significantly, the appropriate chi-square test giving a *P* value between 0.05 and 0.01. The deviation of the backcrosses of *A. cepa* from the expected binominal distribution furnishes additional evidence for the view that the chromosome behavior of the backcross of *A. cepa* is more irregular than that of *A. fistulosum*.

In the plants of amphidiploid × *A. cepa*, one or more chromosome fragments were found in 11.7 per cent of the microspores, whereas in the plants of amphidiploid × *A. fistulosum* fragments were found in only 3.7 per cent. The chi-square test gave a *P* value between the 0.05 and 0.01 levels of significance. This result provides additional evidence that there is more chromosome irregularity in the backcross plants of *A. cepa* than in those of *A. fistulosum*.

Discussion

The behavior of the triploids described in this paper is very different from that of those found by Levan,[13] although the same two species, *A. cepa* and *A. fistulosum*, are involved. Trivalents were rare in the triploids reported here, whereas they were very common in Levan's. This marked difference in behavior probably results from the difference in method of origin of the triploids. The triploids reported here were obtained by backcrossing the parent species to an amphidiploid, whereas the triploids reported by Levan arose in a small second-generation population derived from a diploid first-generation hybrid, possibly from the union of a haploid with an unreduced gamete. Levan[10] found a great deal of irregularity during meiosis in the diploid first-generation hybrids, but considerable pairing with some chiasma formation occurred between the chromosomes of *A. cepa* and *A. fistulosum*, so that some pairs remained united at first metaphase. However, in the amphidiploid reported by Jones and Clarke, the evidence indicates that the chromosomes of *A. cepa* and of *A. fistulosum* remain paired, at least for the most part, with their homologues, forming eight bivalents of *A. cepa* and eight of *A. fistulosum*. It would seem that pairing between homologous chromosomes from the same species takes place whenever possible, and that chromosomes of *A. cepa* remain paired with those of *A. fistulosum* only when another homologue of *A. cepa* is not available, as in the first-generation diploid.

In the triploids obtained from amphidiploid × *A. cepa* and in those from amphidiploid × *A. fistulosum*, in each set of three chromosomes, the two derived originally from one species form a bivalent, and the remaining one derived from the other species separates as a univalent.

Because there is considerable pairing in the first-generation diploid between the chromosomes of *A. cepa* and *A. fistulosum*, and they frequently remain associated as bivalents at first metaphase, it is highly probable that some interchange takes place between them. In this way, chromosomes would arise that are derived partly from one species and partly from the other. Hence in such a triploid a set of three chromosomes no longer consists of a pair derived entirely from one species and a third derived entirely from the other, as in the triploids described in this paper. Trivalents, as found by Levan,[13] may be formed as a consequence of this type of behavior.

Summary

1. An amphidiploid obtained from the cross *A. cepa* L. variety Australian Brown × *A. fistulosum* L. type Nebuka has been backcrossed with both parent species, giving rise to triploids.

2. During meiosis these triploids regularly form eight bivalents and eight univalents.

3. The plants from the backcross

amphidiploid × *A. cepa* can be distinguished from those of amphidiploid × *A. fistulosum* by the difference in the appearance of the bivalents.

4. Greater irregularity during meiosis was found in the plants from amphidiploid × *A. cepa* than in those from amphidiploid × *A. fistulosum*.

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TABLE I. Chromosome counts at first postmeiotic metaphase in triploid Allium plants.

Chromosomes per microspore	Microspores counted	
	Triploid A*	Triploid B†
Number	*Number*	*Number*
8	5	5
9	37	21
10	135	106
11	239	238
12	279	290
13	183	241
14	85	114
15	31	32
16	5	6
Total	999	1,053
P value	Less than 0.01	Between 0.7 and 0.5

*A-Amphidiploid × *A. cepa* (counts from 11 plants).

†B-Amphidiploid × *A. fistulosum* (counts from 10 plants).

◆

Mutation Mink on the New York Market

Advances ranging up to 100 per cent on mutation minks, compared with the previous auction on Feb. 7, were shown at the sale held yesterday for the Mutation Mink Breeders Association by Lampson, Fraser & Huth. The sales approximated $600,000, with quantities offered being about half of those at the previous sale. Turnover was 100 per cent, with the overall price average up 65 per cent.

The offering of 3,521 Royal Koh-I-Nur minks brought a top of $76, and advance of 61½ per cent over the February auction.

Koh-I-Nur minks, of which 3,152 were offered, sold up to $55, a rise of 100 per cent.

The Blufrost minks, numbering 1,675, reached $82, an increase of 62 per cent.

A total of 2,410 miscellaneous mutation minks brought a high of $57, up 44 per cent.

George H. Mayers, manager of Lampson, Fraser & Huth, said that active bidding and higher prices at the sale yesterday reflected highly favorable consumer response to the mutation minks which were offered at the February sale.

He pointed out that at the first auction of the minks, held in 1945, small quantities were offered, which were easily absorbed. When much larger quantities were offered in February, the trade had to test out consumer reaction, which has since developed favorably. —*New York Times*, April 16, 1946.

MUTATIONS OF MINK UNDER DOMESTICATION

W. E. Castle
Department of Genetics, Univ. of Calif., Berkeley, Calif., and
Larry Moore
Mutation Mink Breeder, Suamico, Wis.

IN the first three centuries after North America was discovered by Europeans, the most valued loot which the invaders were able to send back home was the furs taken from wild mammals, beaver, fox, mink, muskrat, and others of less value. The skins were taken by the Indians from animals trapped by them and were delivered to Europeans or half-breed traders in exchange for blankets, arms, tools or trinkets of European manufacture. Great fortunes were in some instances amassed in the fur trade, as for example that of the Astors of New York.

As the farms of settlers encroached upon the domains of the fur bearers, the supply of furs declined and prices increased. This led to more intensive trapping and to a resultant increasing scarcity of fur-bearing animals.

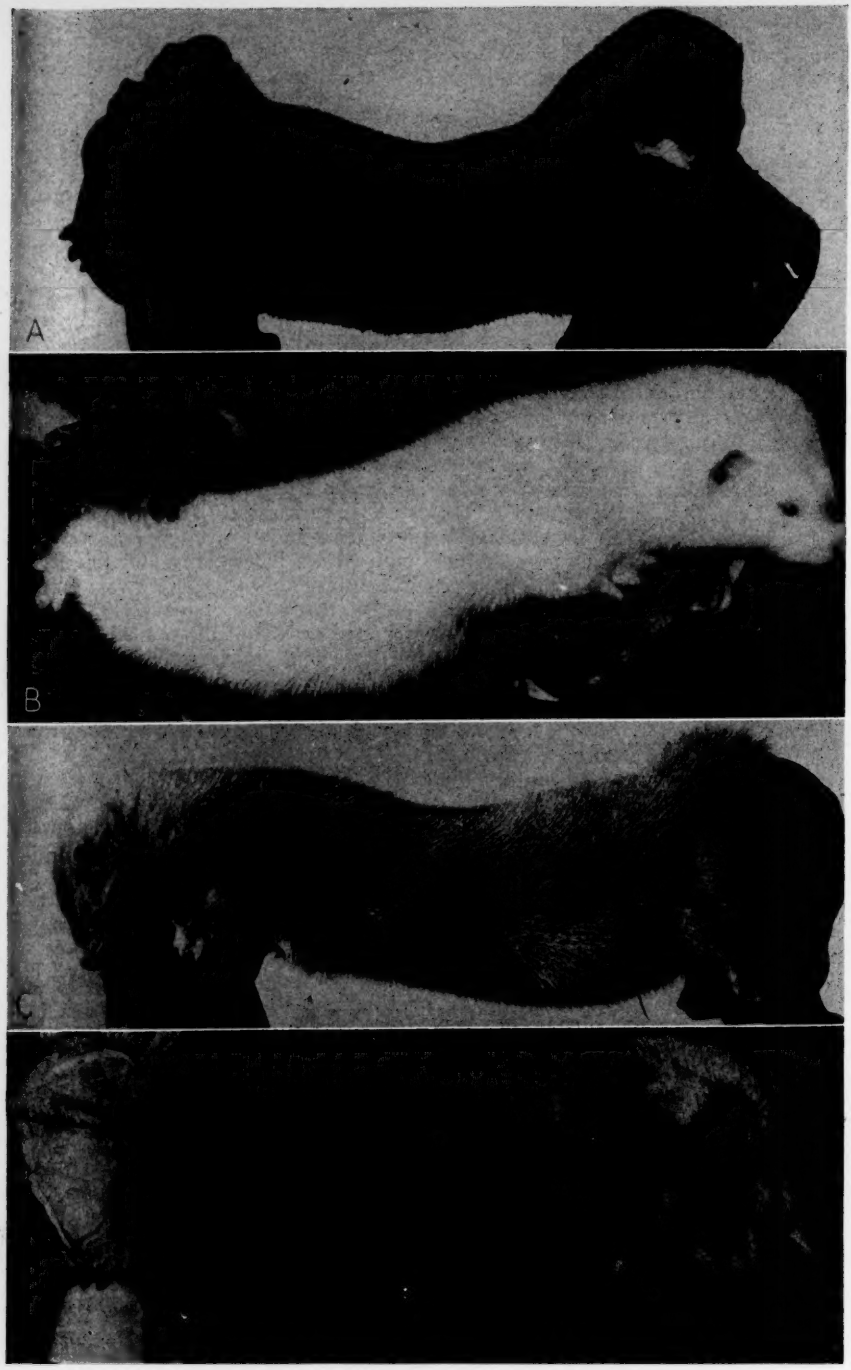
Fur traders were the pioneer explorers of the continent. In search of new sources of fur, they crossed to the Pacific where skins of the prized sea-otter could be had. There they met Russian explorers on a similar errand, as they came down from Alaska as far as Northern California. The sea-otter became practically extinct under merciless and indiscriminate hunting. The Alaskan seal was rescued from a like fate only by an international agreement ratified just in the nick of time between the United States, Great Britain, Japan and Russia to limit the annual take of seals on the Priboloff Islands and to share the profits of the industry. This arrangement has worked well and is still in force except as modified by the results of World War II.

In the interior of the continent, between the Atlantic and the Pacific, the beaver was long the most important source of fur. This led to trapping so intensive as to threaten the source of supply. Beaver became scarce and other furs were sought to replace them, such as fox, marten, mink and muskrat. As these in turn became scarce, efforts have been made in our own time to breed certain species in captivity and thus truly domesticate them. This was accomplished first in the case of the silver fox, a prized variant of the common red fox. A little later mink were domesticated and are now bred extensively in the northern United States, in Canada, and to a lesser extent in northern Europe.

Mink Mutations

A species which undergoes domestication comes under closer observation by man and has its mating controlled by him. Spontaneous variation, when it occurs is more likely to be observed, and if considered desirable can be readily perpetuated by selection and inbreeding of the stock in which the variation has been observed. It is in this way that a considerable number of "mutant mink" varieties have originated in the past 25 years. Some of these have already demonstrated their economic value and others doubtless will in the future. It is the purpose of this paper to describe the more important and better known of these mutant mink varieties, and to explain their genetic peculiarities, so far as at present known. It is quite probable that not all of these mutations have a future in the fur industry. Some are undesirable because of their spotting and lack of uniform pattern. Any interested breeder will please hold this in mind.

To propagate mink in captivity it was found necessary to give them good living conditions, involving secure and sani-



WILD TYPE COLOR AND THREE MUTATIONS

Figure 3

A—Black mink, an example of the original wild color type, perhaps made darker and with a coat of finer texture as a result of selection. *B*—Albino mink ($c^{H}c^{H}$). Note rather coarse texture of fur and smudge of color on nose and tail tip, indicating that it is a feebly pigmented allele of complete albinism. *C*—Silverblu mink (*sb sb*), one of the most valued of mutant races. *D*—Gunmetal blue (*gg*), a mutation darker than Silverblu and distinct from it genetically.

tary cages, an adequate and nourishing diet, and to take precautions against injury by fighting. Pelts also had to be taken, when prime, at the proper time of the year. All these practical problems have now been solved satisfactorily by the cooperative efforts of a large number of ingenious and observant mink breeders who have shared each other's practical discoveries in technique and in errors to be avoided. Into such matters we shall not enter here.

To produce mink fur from captive animals, it was necessary to obtain superior foundation stock. This consisted of animals caught in the wild having good constitution, and with good texture and color of the fur. This matter was given much careful attention by the pioneer breeders of mink, because they had to match in their product the quality of wild caught fur or take a lower price for their product. To their credit be it said they succeeded remarkably well, taking their breeding stock largely from northern areas, Alaska and northern Quebec, where large robust animals were found with a desirable dark and lustrous coat. In the early foundation stock, particularly that of Alaskan origin in which the body size was greater than that of the Quebec stock, the texture of the fur was inclined to be coarse, but this was improved by intercrossing of Quebec with Yukon stock followed by careful selection for a dark coat with fur of fine texture, so that now cage bred mink of the wild type of color command top market prices.

Basic Genes

We may assume that wild mink have the basic *A B C* dominant color genes of wild mammals in general. Let us consider the action of these genes in determining coat color.

(1) *C* is the symbol of the albino series gene, whose activity is essential to the production of any color whatever in the coat. If as the result of recessive mutation, the color gene *C* has its activity restricted or entirely suppressed, there results an albino of some sort. A true and complete albino, wholly devoid of pigment in the coat, skin and eyes, is given the genetic symbol c.

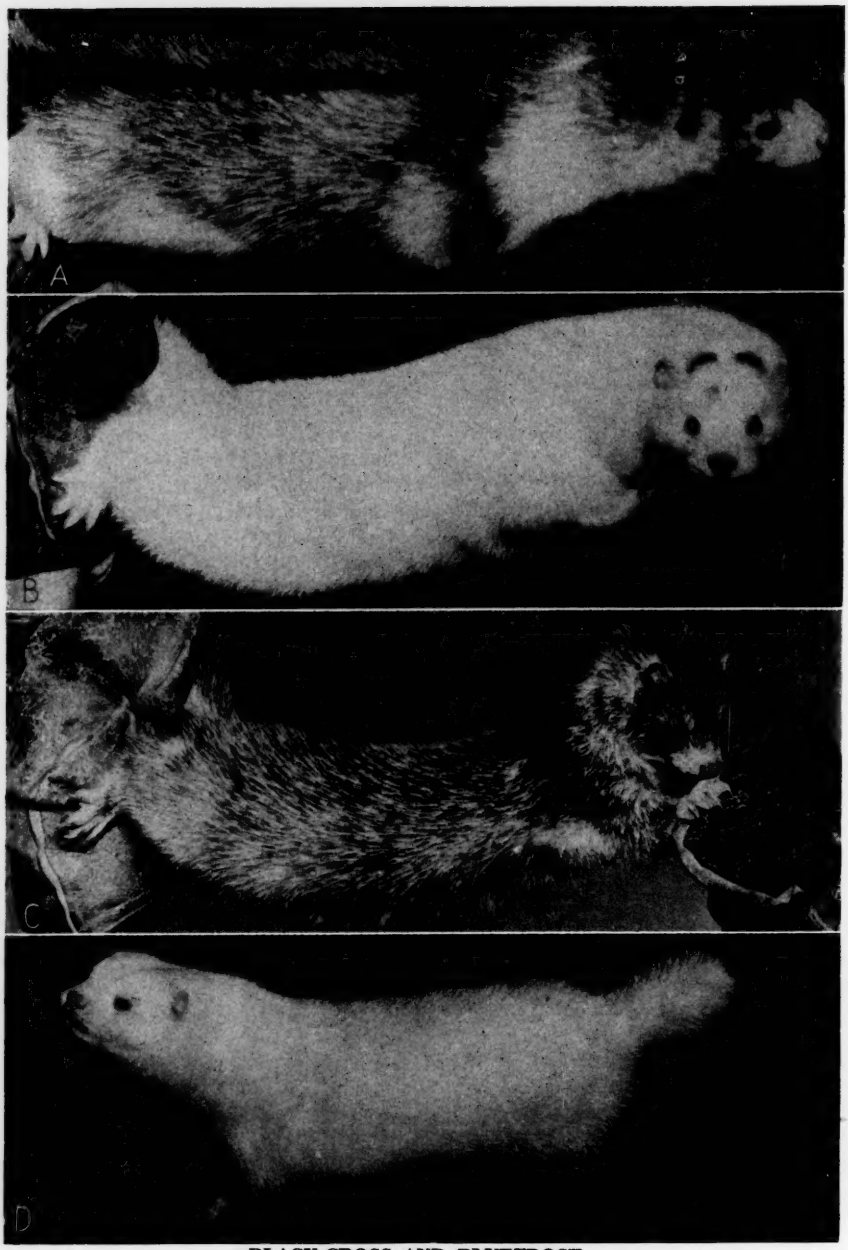
A type of albino in which pigment is developed sparingly in eyes and coat is found in the Himalayan rabbit and in similarly pigmented varieties of other rodents as well as in the Siamese cat. This albino allele has been designated c^{H}. It is recessive to *C* but dominant over c in crosses.

A higher grade of albino allele, more heavily pigmented than the Himalayan albino but lighter in color than the wild type is represented by the Chinchilla rabbit and by similar pale coated varieties of other rodents.

Generally, each grade of albino allele, is dominant over all less heavily pigmented alleles, but recessive to all more heavily pigmented ones, as well as to full color. Hybrids between different albino alleles are to a certain extent intermediate between their parents in depth of color, though nearer to the darker type. We shall see that albino mink, so far as we have been able to inspect them, are of the Himalayan type with smudges of color on the extremities. In this they resemble the albino guinea pig.

(2) A second basic color gene of mammals is a gene for black color (symbol *B*) which determines black pigment as the end product of oxidation of a color producing substance in hair, skin and eyes. As an alternative end product, when a recessive mutation *b* is substituted for *B*, a chocolate brown pigment is formed instead of black. This brown mutation is known to have occurred in all the common laboratory rodents, such as rats, mice, rabbits and guinea pigs, as well as in dogs and horses. We find that it is present in the mutant race of mink known as Pastel or Blond. Probably in albino mink, as in guinea-pigs and rabbits, the smudging would become less in combinations which include the pastel mutation in homozygous state (*bb*).

(3) A third basic color gene of mammals determines the wild type of coat pattern and is expressed by the symbol *A*, abbreviation for agouti, a South American animal having this coat pat-



BLACK-CROSS AND BLUEFROST

Figure 4

A—Koh-i-nur or Black-cross, heterozygous (*Kk*). *B*—Koh-i-nur or Black-cross, homozygous (*KK*). *C*—Blufrost or Silver Sable, heterozygous (*Ff*). *D*—Dominant white, a combination type (*KKFf*), homozygous for *K*, heterozygous for *F*.

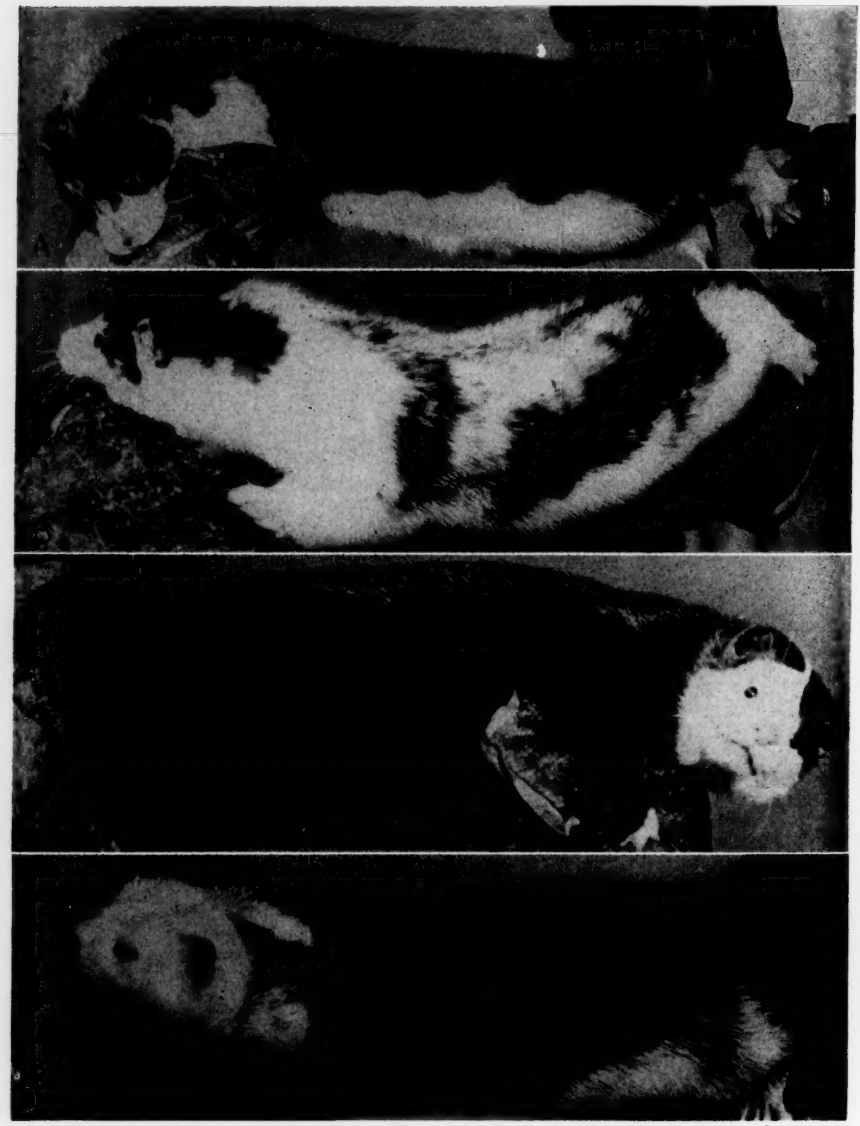
tern excellently developed. An example more familiar is the wild gray rabbit which has, in its dorsal coat, hairs tipped with black, but with a subapical band of yellow and below that a dull leaden black base. All ventral surfaces, including the neck, belly, and underside of the tail, are white or whitish, devoid of black-tipped banded hairs. By recessive mutation from *A* to *a*, the wild or agouti type of coat pattern is completely lost, the coat becoming uniformly black all over, though with less heavily pigmented ventral surfaces.

A less extreme mutation of the wild coat pattern is represented in the black-and-tan variety of the rabbit and some other rodents. This allele is designated a^t. The black-and-tan rabbit has a back of uniform black, with a whitish belly, but sides sparingly furnished with black-tipped banded hairs of the agouti type. Here the wild type of coat pattern has been only partially lost, whereas in the black variety it is entirely lacking.

The wild mink has no very obvious coat pattern. The dorsal surfaces are more heavily pigmented than the ventral surfaces but there is no distinct agouti banding of the hair. What possibly corresponds with the yellow subapical band of an agouti pattern is found in the very extended reddish markings of the hair, obscuring both a distinct black tip and a leaden black or bluish base. Grinnell[1] describes the coloration of wild California mink as follows: "General coloration of body, including legs and feet, cinnamon-brown, only slightly paler beneath than above; darkest, but not blackening, on top of head and down middle of back; still darker on tail, becoming nearly black toward tip. A small white spot at tip of chin." We know that the early breeders of mink in captivity sought to get as dark a coat as possible, avoiding the red type. Whether in the course of their selections animals of a genuine uniform non-agouti black type (*aa*) were secured it is impossible at present to state. Certainly such improved mutant types as the Silverblu show no evidence of a genuine agouti wild pattern. It is problematical whether as regards coat pattern they are *A* or *a* in genetic constitution, so we shall not attempt to classify the mutant varieties in this regard.

We do know that in wild mink there is a certain amount of ventral white spotting. In the words of Grinnell, "small irregular white markings are usually present on chin and middle of belly." The extent of the white markings is subject to mutational change, as we shall see. In the dominant mutants, Koh-i-nur and Blufrost, the ventral markings are greatly extended, especially in the former, in which the white area extends upward on the sides of the body, on to the nose and the underside of the tail. In certain mutants, less well known but also dominant, Baldy and Whiteface, the extension upward of the white areas is chiefly in the head region. In Blackout, it seems to occur along the entire length of the body, as in Koh-i-nur.

The pioneer breeding of mink in captivity was directed toward the production of fur like that of the best skins of the trappers, which came chiefly from northern Quebec and was very dark in color. Selection was made accordingly in the cage-bred stock for darkness of fur and fineness of texture. Improvement was secured in both particulars. As mink were reared in increasing numbers, individual freak animals, sports or mutations, began to appear and observing breeders gifted with curiosity put them aside or bred them to their near relatives to see what would be the outcome. Thus mutant varieties, mostly lighter in color than the original wild type, had their origin. We shall describe the more important and better known of these. Close-up pictures of live animals, though not artistically posed, will, we hope, help to give an idea of the character of the fur in these mutants. Numerous keen-eyed breeders deserve to share in the credit for the detection, the propagation and the improvement of the mutant varieties. We shall not attempt to assign credit to them individually, lest thereby we seem to disparage the contribution of others, and particularly because Hodgson[2] in the *Mink Book* has



BLACKOUT AND BALDY

Figure 5

A—Blackout, a dominant mutation resembling Koh-i-nur, of which it is possibly an allele. Heterozygous individual. *B*—Homozygous Blackout individual. Note resemblance to Blackcross. *C*—Baldy, a dominant mutation in which the original white throat spot has extended upward over the entire head. Heterozygous individual. *D*—An F_1 Baldy $\times$ Koh-i-nur individual showing effects of both dominant mutations.

gone into the matter in considerable detail.

Recessive Mink Mutations

1. Albino

The albino mink has pink eyes and white fur, which however is not a clear white all over the body, as is the coat of the ordinary white rabbit. It has a smudge of color on the nose and the tail. Since in this it resembles the Himalayan rabbit, its mutant gene may be classified as an albino allele, similar to that of the Himalayan rabbit, with a genetic formula of $c^{H}c^{H}$. It is recessive in crosses with the normal colored type. Some of the original mutant albinos seem to have been rather coarse in texture of fur, a fault capable of correction by crossing with the more improved colored types. See Figure 3*B*.

2. Blond

A strikingly beautiful color mutation of mink recessive in heredity is known as Pastel or Blond, symbol *b*. It corresponds with the cinnamon mutation of rats, mice, rabbits and guinea-pigs. In this mutation, black which is the predominant dark pigment in the wild type of coat, is everywhere throughout the body replaced with brown pigment. So far as black pigment is concerned the wild mink is homozygous *BB* in formula, the mutant type Pastel being *bb*, homozygous brown. The brown coat of Pastel mink has a slightly purplish tint, when in full prime. See Figure 6*A*.

3. Silverblu

Silverblu is the name adopted by the Mink Breeders Association for one of the most valued and beautiful of the recessive mutant varieties. For it we shall use the genetic symbol *sb*. It is much lighter in shade than wild mink and bluish in color both of guard hairs and of underfur. See Figure 3*C*.

4. Imperial Platinum

Imperial Platinum (symbol *p*) is the name given by its discoverer to a mutation similar in appearance to Silverblu, but entirely distinct in origin and genetic behavior. When it is crossed with Silverblu, young are produced of the normal wild colored type.

5. Gunmetal Blue

Gunmetal blue (symbol *g*) is the name given to another pale-coated recessive mutation of recent origin. In its blue color it is darker than Silverblu, but like it, shows "a pleasing contrast" in tone of color between guard hairs and underfur. See Figure 3*D*.

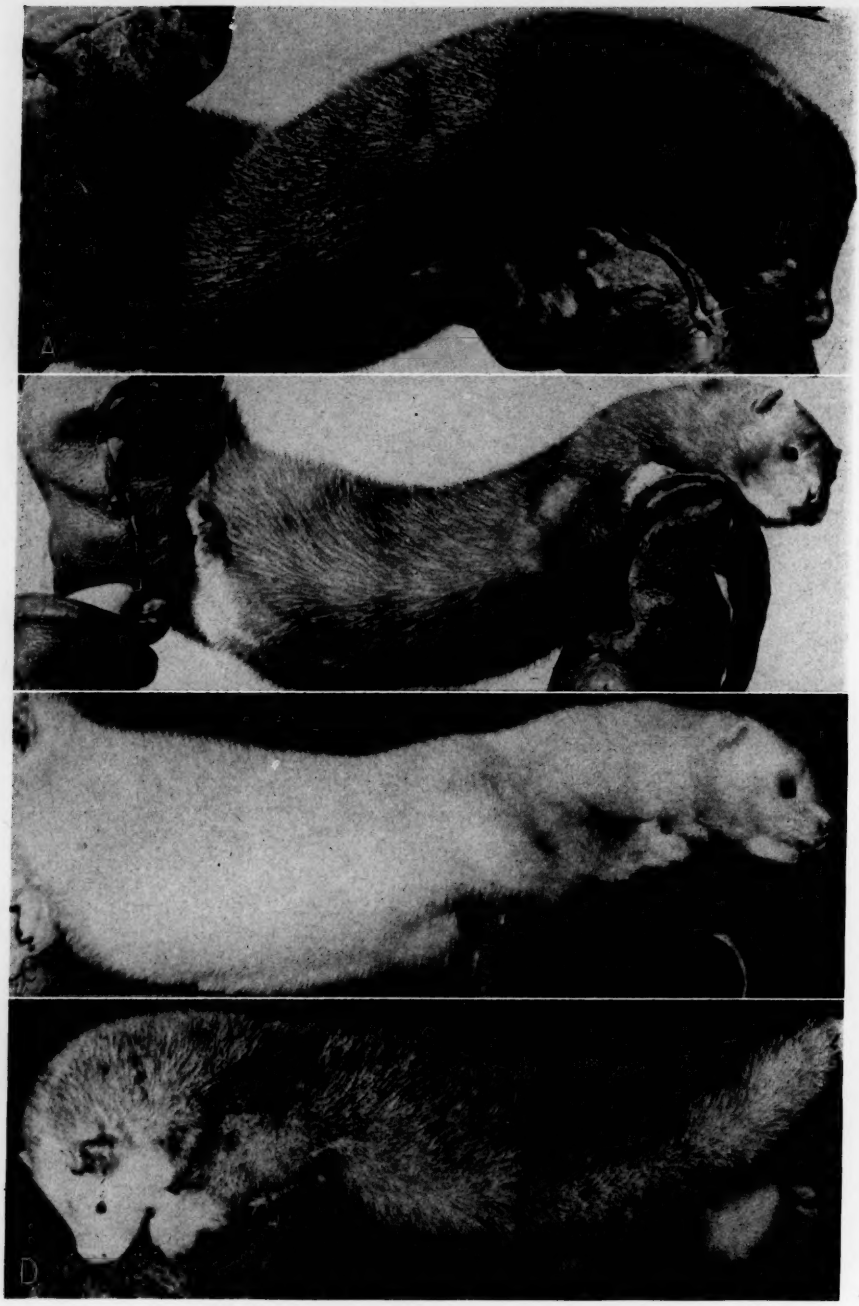
Dominant Mink Mutations

6. Koh-i-nur

A striking mutation in coat pattern originally called Black Cross was later renamed by the Mink Breeders Association Koh-i-nur (symbol *K*). See Figure 4*A*. It greatly reduces the pigmentation of the animal both by extension upward of the ventral white area and by depigmentation elsewhere of the underfur and to a lesser extent of the guard hairs also. The underfur becomes practically white all over the body and the pigmentation of the guard hairs is restricted to a dark stripe along the middle of the back and a transverse bar across the shoulders (the black cross). Lateral to the back stripe only part of the guard hairs are black and they are gradually replaced, progressing outward, by hairs entirely white. Such is the effect of the mutant gene *K* in single dose, i.e. in heterozygotes. In homozygotes (*KK*) its depigmentation effects are much greater. The back stripe becomes narrow and interrupted or may disappear altogether, leaving pigmented spots only on the top of the head and at the base of the tail, as in the animal shown in Figure 4*B*.

7. Silver Sable (Blufrost)

Silver sable was the name originally given to a dominant mutation later renamed Blufrost (symbol *F*). See Figure 4*C*. In heterozygotes (*Ff*) this mutation effects marked reduction in the amount of pigment in the coat, in this respect resembling Koh-i-nur. But the manner of pigment reduction is very different in the case of Blufrost, where it consists primarily in the silvering of the guard hairs and a lightening in color of



RECESSIVE PASTEL

Figure 6

A—Pastel (*bb*), a recessive mutation containing brown pigment but no black. The photograph in black and white gives a wrong idea of the color which is really brown not black. *B*—A combined Blufrost Pastel individual (*Ff bb*), heterozygous for *F*, homozygous for *b*, known as "Breath of Spring" Pastel, or Silvered Pastel. *C*—A combined Blufrost Silverblu individual (*Ff sb sb*), heterozygous for *F*, homozygous for *sb*, known as "Breath of Spring" Silverblu. *D*—An F_1 Blufrost × Baldy individual, heterozygous for both dominant mutations.

the underfur. The amount of silvering in the guard hairs varies widely and is subject to control by selection. It ranges from a condition in which the coat is heavily silvered with pure white guard hairs to one in which very few guard hairs are silvered. The color of the guard hairs is also variable, ranging from light brown to intense black, the latter condition being considered more desirable as increasing the contrast with the underfur, which is a clear light blue in color. Blufrost, like Koh-i-nur, has a tendency to enlarge the ventral white areas but to much less extent than the latter.

Its effects in a homozygous state are unknown, as the existence of a living homozygote has not as yet been demonstrated. Possibly that condition is lethal. Blufrost animals mated to each other produce litters of reduced size and no silvered individuals obtained from such matings have been shown to be homozygous by failing to produce non-silvered young in outcrosses.

8. Dominant White

Special mention should be made of a *dominant white variety*, having colored eyes (Figure 4*D*), which may be created by combination of Koh-i-nur with Blufrost. A cross of these two, if both parents are heterozygous, will produce in F_1 a certain number of individuals (theoretically 25%) which are double heterozygotes (*KkFf*). They can be recognized as being the whitest individuals in the group, in appearance resembling homozygous Koh-i-nurs, with a very narrow back stripe or none at all. but with head and tail spots only. If such a double heterozygote is backcrossed to a Koh-i-nur, individuals will be produced which are *KKFf* in constitution, homozygous for *K*, heterozygous for *F*. Such animals will be practically all white with "not over 12 dark hairs." In outcrosses with the normal colored type, they should produce four groups of young numerically equal, which as regards the dominant genes *K* and *F* should be: (1) *KkFf* (white like other F_1 double heterozygotes), (2) *Kk* (ordinary Koh-i-nur, (3) *Ff* (ordinary Blufrost), and (4) normal dark.

If dominant white individuals (*KK Ff*) are mated with each other, it is to be expected that two-thirds of the young will be dominant white like their parents, the other third being homozygous Koh-i-nurs (*KK*). A theoretical fourth class (*KKFF*) is not to be expected if, as assumed, homozygous Blufrost is lethal.

9. Baldy

A dominant coat pattern mutation known as Baldy (Figure 5*C*) has a white head and generally paler coat. The underfur is light blue slate.

10. Blackout

Blackout (Figures 5*A* and 5*B*) is an imperfectly tested dominant mutation, in which the coat is intensely black and slightly silvered. It has a large amount of white on belly, feet and nose, with white extended upward in the neck region. Possibly it is an allele of Koh-i-nur.

11. Combination

A promising combination type of dominant Blufrost with recessive Silverblu (*Ff sb sb*) is lighter in color than Silverblu and possibly of a clearer blue color. It is known by the romantic name of "Breath of Spring Silverblu." See Figure 6*C*.

A similar combination type of dominant Blufrost with recessive Pastel (*Ff bb*) is lighter in color than Pastel, with a bluish cast in underfur and guard hairs. It is known as "Breath of Spring Pastel." See Figure 6*B*.

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ANIMAL BREEDING AS SCIENCE AND ART

A Review

ANIMAL breeding is a business; and, therefore, economic considerations of the value and availability of time and materials loom larger in it than they do in the investigation of a purely scientific problem. The work must go on, and decisions must be made in many cases where there is not yet enough evidence to show with certainty what the result will be. So states the author in his preface to *Animal Breeding Plans*.* He continues by pointing out that the scientist might retire to his laboratory and design an experiment which would in due time reveal the truth in such cases. But the man engaged in breeding animals cannot wait. Without being certain what would result from each possible alternative, he must decide whether to cull an animal or keep it, and if it is kept, how to mate it. With these needs of the breeder in mind, the author has sought to state the most probable truth concerning questions which may guide his decisions, even in cases where genetic knowledge is not yet complete enough to establish closely the limits of truth.

The book is so organized that material falls under seven major headings. In the first section the student is given some of the background of animal breeding. A brief outline is given of the origin and domestication of animals, the consequences of domestication and the development of pedigree breeding. This is followed by a historical sketch of animal breeding methods in the United States, four stages of development being recognized: The pioneer period when the kind of livestock was not of much importance; a periol of developing local races and a little experimenting with introduced breeds; a period of extensive experimenting with introduced breeds and widespread expansion of organized pure breeding; and the present period in which the chief interest is in breed improvement. This interest is stimulated by competition among breeds and by the need for keeping purebreds distinctly ahead of grades of the same breed in order to maintain a demand for purebred sires. There is also a review of the work of breed associations, from which the author concludes:

> Most of the breed *improvement* has to be done by the breeder himself. . . . Breed associations must remain more or less responsive to the opinions of the majority of the breeders and therefore cannot be expected to do much pioneering or testing of new and unpopular ideas. This will have to be done by venturesome breeders or by public research institutions. . . . The associations' part is on the whole the conservative one of helping hold whatever average merit the breed has already attained. . . .

Genetic Principles

The second section is devoted to basic principles and includes discussions of Mendelian inheritance, the genetic basis of variation, the relative importance of heredity and environment, the nature of the differences between breed populations and other populations with which the breeder may deal, and an outline of the means available for controlling animal inheritance.

The success of any improvement program depends to a large extent upon the ability of the breeder to select, as parents of the next generation, those animals that are really superior. The third section of this book deals with problems of selection. Two chapters deal with the effects of selection upon the genetic and phenotypic makeups of populations. These are followed by seven chapters in which the yardsticks available for use in selection are discussed. Attention is given to lifetime averages of production, pedigree estimates, progeny tests, selective registration, type and production records, breed type, and the show ring. The needs for adequate evaluation of existing methods of selection and for the development of improved methods of

Lush, Jay L. *Animal Breeding Plans.* Edition 3. Pp. VIII, 443. $3.00. The Collegiate Press, Inc., Ames, Iowa. 1945.

selecting the superior animals constitute two of the most important problems in animal breeding. In the practice of the art of animal breeding, many methods have been evolved, but little has been done to determine if they are based on sound scientific facts. The discussions in the chapters noted above give a candid evaluation of what the breeder can expect from the various procedures discussed. The techniques of selection and the problems involved in evaluating and improving them are too many and too varied to deal with in a review. The author of a text is also limited in space, and in this section only a few of the many techniques for evaluating specific characters are mentioned. Attention is given primarily to the basic principles, which of course apply regardless of the type of livestock or the specific characters one is attempting to improve, and regardless of the method of breeding employed.

Relationship

In the fourth section, attention is given to breeding plans which are based on relationship. An indication of the content and thought in this section can best be given by a few quotations from the author's chapter summaries:

Likeness between relatives—degrees of relationship:

Relationship measures the probability that individuals will be alike *in their genes.* Their actual outward likeness depends also upon how much the traits being measured are affected by environment, dominance, and epistasis, upon the extent to which their environments were correlated, and upon whether their ancestors were mated like to like.

The most important practical use of relationships is in predicting the most probable merit of unknown or perhaps even unborn individuals from the merit of their known relatives.

The consequences and measurement of inbreeding:

Inbreeding is the most powerful tool the breeder has for establishing uniform families or strains which are distinct from each other. . . . .

The danger of intense inbreeding is that it will make undesired genes homozygous at so rapid a rate that it will be impossible to discard all individuals homozygous for them. The lowered sale value of the defectives uncovered by the inbreeding will cause some prepotency of those which are not defective. . . .

It seems reasonably certain that more opportunities for breed improvement are lost by not inbreeding when inbreeding would be advisable than are lost by too much inbreeding. . . .

Prepotency:

The genetic basis for prepotency is the degree of homozygosity of the animal and whether its genes are prevailingly dominant or recessive. To a small extent linkage and epistasis may play some part.

Almost the only control the breeder has over prepotency is the extent to which he builds homozygosis into his animals by inbreeding.

Linebreeding:

Linebreeding is a form of inbreeding directed toward keeping the offspring closely related to a highly admired ancestor. All inbreeding not necessary for holding this relationship high is avoided as far as possible. Hence, the intensity of the inbreeding is usually moderate in linebreeding systems. Relationship to a chosen ancestor is the main feature which distinguishes linebreeding from other forms of inbreeding.

The family structure of populations:

The term "family" in animal breeding implies a group of animals related to each other, but usage varies widely as to how close that relationship must be before two individuals are considered as members of the same family. Often the definition of family is vague and variable, even in a single discussion.

If females are consistently mated to males of the same family as their own, such linebreeding tends to make and keep families distinct from each other. If the linebreeding continues long or becomes intense, such a linebred family tends to become a breed within a breed. Such distinct families within pure breeds have been formed only occasionally.

Family selection is most helpful for characteristics for which heritability is low and family members do not resemble each other for any reason other than their genetic relationship.

Blood-lines:

'Blood-line" is an elastic term used sometimes as synonymous with family, sometimes as a substitute for relationship, and sometimes to describe vaguely a breeding system.

Outbreeding systems:

Outbreeding generally leads to individual excellence but low breeding worth.

Outbreeding systems hamper progress in further improvement of a breed because they destroy families by constantly crossing together any which start to develop. Thus they keep the breed temporarily more uniform than if outbreeding were not practiced.

Crossbreeding is a special form of outbreeding where the parents belong to different

breeds. It generally results in increased size, vitality and fertility; but the amount of this increase is variable in different crosses. The economy of crossbreeding depends upon whether the increase in these things is more than enough to balance the possible confusion and increase in cost of replacements under a crossbreeding system.

Grading is the continued use generation after generation of males of one pure breed on an original foundation of another breed or of no particular breed. Grading is the most economical way of lifting the commercial stock rapidly toward the level of the purebreds.

In the fifth section attention is given to breeding plans which are based on somatic likenesses and differences. Much has been written about the importance of mating like to like. The author points out that such matings have almost no effect on homozygosis except in very simple and rare circumstances. Mating of like to like comes near to the full limits of its effects within a very few generations after it is begun, and such effects disappear almost at once when random mating is resumed. Mating of unlikes is useful in maintaining uniformity in the group until the average type or some other intermediate type can be fixed by close inbreeding. It may also be useful in correcting defects wherever the ideal is intermediate and some animals are too extreme in both directions.

Some additional topics concerning breeding plans are discussed in the sixth section. These inclule the relative importance of the sire and dam, the registering of high grades, sire indexes, bull assocations, and community breeding. In discussing the posibility of community breeding, the author points out that most of our breeds were formed originally by more or less definite community breeding. Occasional examples of such community breeding have occurred in America, but this has not yet become the general practice in any nationally important breed.

Several topics relating to reproduction are discussed in the seventh section, including masculinity and feminity, hermaphrodism and other abnormalities pertaining to sex, gestation periods, sex ratios, and fertility and breeding efficiency. Most of these discussions are developed from the genetic aspect and very little attentoin is given to the many important problems of physiology of reproduction with which the breeder must deal if he is to obtain maximum reproductive efficiency.

In a concluding chapter, attention is given to problems that individual breeders encounter when planning their operations, to an ideal system for rapid breed improvement, to unsolved problems, and to opportunities in the animal breeding field. The system proposed for rapid improvement of a breed is the dividing of the breed into small groups, practicing mild linebreeding in each of these groups, and making occasional exchanges of stock between groups when this seems desirable, both breeding methods being accompanied at all times by intense selection.

The general pattern of the book is similar to the earlier editions, but it has been expanded considerably in size. It has grown out of sixteen years of teaching animal breeding to college students who have had courses in genetics, embryology, anatomy and physiology of farm animals, herdbook study, history of breeds, and stock judging. Obviously, it is intended for the advanced stulent, and is not so well suited for use in animal breeding courses in which the student is not already well grounded in genetics, physiology and related subjects. This would be particularly true if the instructor were not well grounded in the statistical aspects of livestock genetics. In addition to the advanced undergraduate student, *Avimal Breeding Plans* should be useful to graduate students in animal breeding, to animal husbandry staff members that deal in any way with breeding problems, and to breeders who wish to approach their genetic breeding problems in the light of the best information available for solving these problems. Since it is best adapted to use by advanced workers, its value would be enhanced by a more complete documentation of pertinent reference material.

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GENETICS OF SESAME

III. "Open Sesame" and Mottled Leaf

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IN one of the best known stories of the Arabian Nights, Ali Baba uses the magic words "Open Sesame" to gain access to the cave of the Forty Thieves. Similarly, he uses "Close Sesame" to put the huge door back into place. These words are significant in that they are based on the extreme dehiscence of the seed pod of sesame (*Sesamum indicum* L.), one of the oldest cultivated oil crops in the world. This crop is still grown extensively in China and India and the seed constitutes an important part of the diet. The dehiscence of the seed pod is useful to those people in that the crop may be threshed by simply inverting the mature plant and letting the seeds rain out onto a blanket. No machinery is necessary.

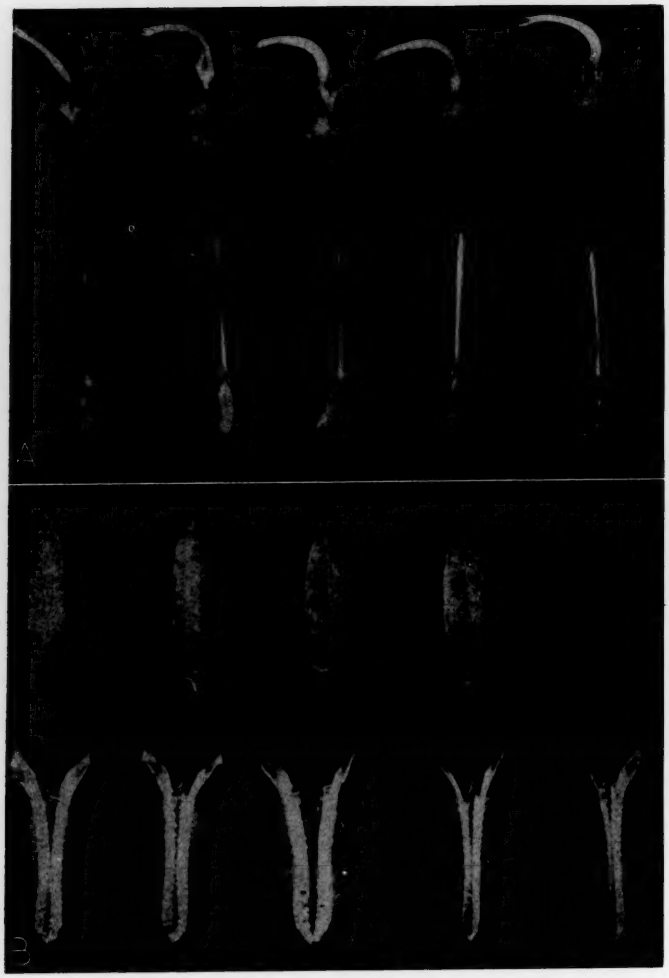
But in the Western Hemisphere where mechanization is the keynote to crop production, this same dehiscence of the seed pod has been the principal obstacle to the widespread acceptance of sesame as a commercial crop. The harvest problem is further complicated by lack of uniformity in ripening. The plant is of indeterminate growth and the seed pods at the base of the plant may be opening and losing their seeds while flowers are still forming in the upper part of the same plant. To this may be added the difficulty caused by having the plants in the sandy areas of the field mature in advance of those in the more moist spots.

When the sesame breeding program was initiated in Venezuela in 1940, the problem of mechanical harvesting was given priority. It was attacked from three angles: 1, selection of plants more uniform in ripening from base to top, 2, bulk selection of plants of the same date of maturity, and 3, search for a chance mutation into an indehiscent or semi-indehiscent type. Rapid progress was made in achieving the first two objectives. Among the many different varieties collected in various countries, plants possessing the desired characteristics occurred and were isolated by selection. But "Close Sesame" proved to be more difficult for us to achieve than it was for Ali Baba.

Hundreds of thousands of individual plants in commercial plantings were examined in an attempt to find the desired mutant. Young plants were treated with colchicine[1] in the hope that the tetraploid form would be indehiscent. It was not. Another possibility was that a suitable combination of complementary factors could be obtained by hybridization of the many varieties.

Finally, in a propagation plot of the F_5 generation of a hybrid between *Criollo*, from Venezuela, and *Selection 5*, an isolated plant found near an irrigation ditch in the same country, one plant differed from all the others in that the leaves were cupped upwards (Figure 8). When this plant matured, the seed pods remained closed (Figure 7*B*) instead of opening like ordinary sesame: it was indehiscent.[2] Seeds from this plant were sown immediately after harvest (November 1943) and most of the seedlings had cupped leaves like the original plant. The few "normal" plants were to be expected from an open-pollinated indehiscent plant. In this population, hand pollinations were made between plants of the cupped-leaf type and *Selection 43-71*.

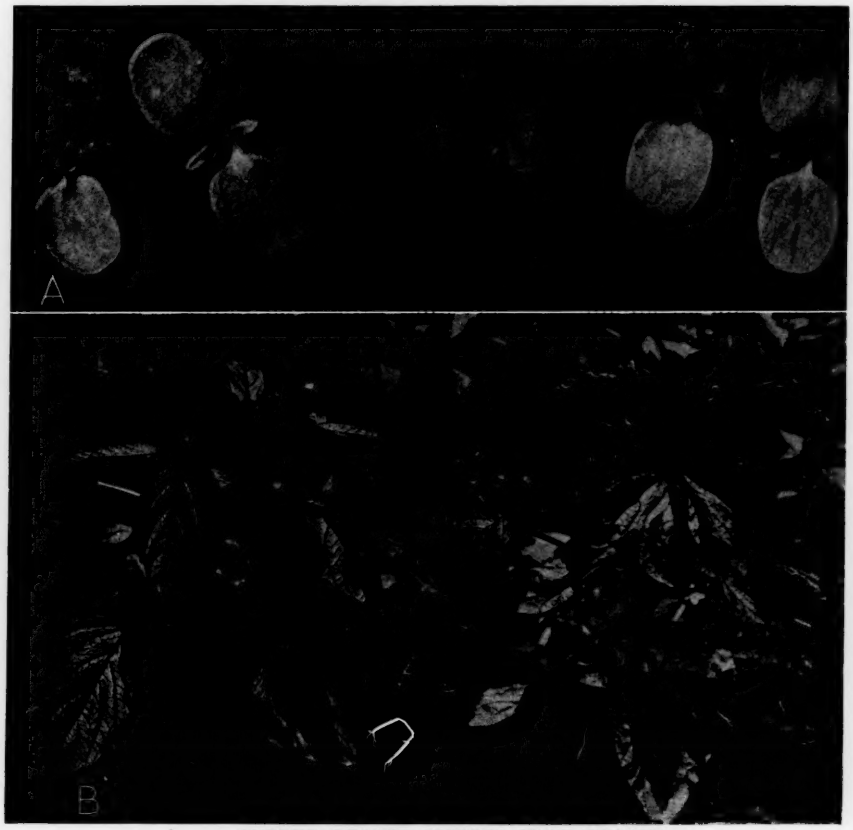
The F_1 hybrid from this cross was dehiscent like ordinary sesame. In the F_2 generation it was possible to classify the seedlings according to whether the cotyledons and the leaves were cupped up-



STYLES AND CAPSULES OF "OPEN" AND "CLOSED" SESAME

Figure 7

A—Curved styles (upper row) are the chief cause of semi-sterility in the closed mutant. In the lower row straight styles of normal sesame are shown. *B*—Ordinary dehiscent "open" sesame pods are shown in the lower row. Under field conditions these plants lose nearly 100 per cent of their seed. Seed pods of "closed" sesame (upper row) retain their seed even when mature.



CUPPED LEAVES VERSUS FLAT LEAVES

Figure 8

A—F_2 seedlings of a cross between indehiscent and dehiscent sesame. Left to right: Plants 2 and 5 have flat cotyledons like ordinary sesame; plants 1, 3 and 4 have cupped cotyledons. These will develop into mature plants with abnormal leaves, like the plant at the right in *B*. An "open" sesame plant with flat leaves is shown at the left.

ward like the indehiscent parent, or horizontal like common sesame (Figure 8*A*). Data from part of this second generation are given in Table I.

TABLE I. Segregation of dehiscent and indehiscent plants in the F_2 generation of a cross between an indehiscent mutant and selection 43-71.

Type of Seedling Leaf	Number of Individuals		
	Observed	Calculated (3:1)	X^2
Cupped	556	586	
Flat	1789	1759	
Total	2345	2345	0.0172

These data indicate that the indehiscent mutant is inherited as a simple Mendelian recessive. The symbol *Id-id* is suggested for this contrasting pair of factors.

Unfortunately, this particular indehiscent form of sesame, in its present condition, is not the final answer to the harvesting problem. The gene *id,* in the homozygous form, has multiple effects on the development of the plant. In addition to the cupped effect due to leaf-

LEAF-LIKE ENATIONS

Figure 9

Leaf-like enations occur on the vental surface of the cupped leaves of indehiscent plants.

like enations in the axils of the veins on the ventral surface of the leaves (Figure 9), there is a still more detrimental secondary character. Indehiscent plants have a curved style (Figure *7A*)). This serves to separate the stigma from the anthers by a short distance, thereby causing approximately 30 per cent sterility. But there is some hope for the situation, as in the second generation of the above-mentioned cross some plants were isolated with less enations and nearly straight styles, with a corresponding increase in fertility.

Mottled Leaf

In one F_3 progeny of a cross between the variety *White Sesame India* and *Selection 5*, approximately 25 per cent (Table II) of the plants had yellow blotches in the upper leaves. These yellow areas with irregular boundaries were numerous in the leaves of some of the segregates, but few in others. By standing over each plant and looking straight down on it, even the most faint trace of yellow tissues could be seen and the plant properly classified. The loss of chlorophyll in the mutants was insufficient to affect the normal growth of the plants. Furthermore, the blotched areas were free from disease and necrosis. The ease of classification and normal vigor of the plants make this a useful character for genetical studies of sesame.

The F_2 distribution of green leaved and mottled-leaved plants is given in Table II. These data indicate simple inheritance. The symbol *M-m* is suggested for this contrasting pair of characters.

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TABLE II. Segregation of mottled and green leaves in a cross of *White Sesame India* and *Selection 5*.

	Number of Individuals		
Leaf Color	Observed	Calculated (3:1)	X^2
Mottled	38	39.75	
Green	121	119.25	
Total	159	159.00	0.01

SYMPOSIA ON QUANTITATIVE BIOLOGY

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As part of its policy of fostering a closer relation between biology and the other basic sciences.. The Biological Laboratory of the Long Island Biological Association, Cold Spring Harbor, Long Island, New York, invites each summer a group actively interested in a specific aspect of quantitative biology, or in methods and theories applicable to it, to take part in a symposium at the Laboratory. The aim is that every important aspect of a given subject should be adequately represented, from the physical and chemical, as well as from the biological, point of view.

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HEREDITARY LEGG-CALVE-PERTHES' DISEASE

F. E. Stephens and James P. Kerby*

OSTEOCHONDRITIS deformans coxae juvenilis is a condition which was first described by Legg of Boston in 1909; by Waldenstorm in Sweden the same year; by Calve and by Perthes in 1910. Since then many articles have appeared in the literature. Most of these describe the condition in such detail that no attempt is made here to discuss the minutiae of the pathology, X-ray appearance or symptomatology.

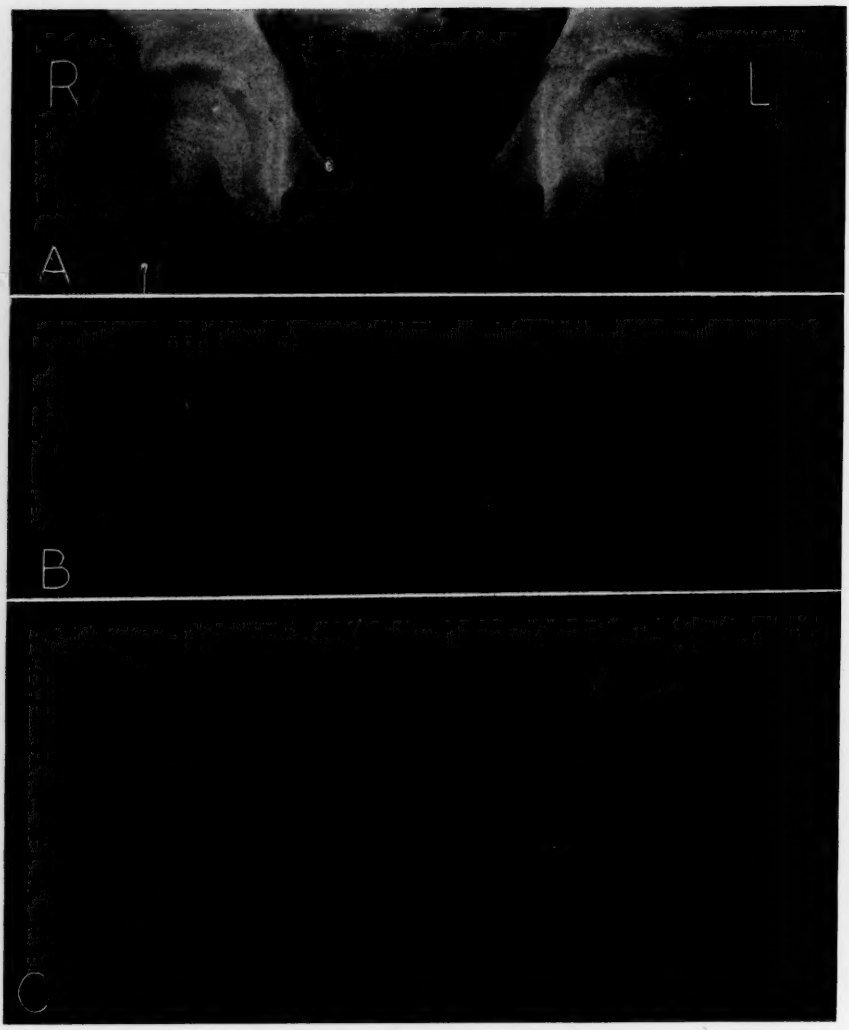
Description

Briefly, it is a condition occurring in the head and neck of the femur. The name is a misnomer, if viewed from the standpoint of "osteochondritis." It is simply an avascular necrosis of the head of the femur. Dislocation of the hip, transcervical fracture of the neck of the femur in children, and epiphyseolysis all produce clinical and radiographic signs which are essentially the same as those produced by this condition. This is particularly true of the X-ray appearance when seen years after the condition has become quiet. Its frequent occurrence has been explained on the basis of the vulnerability to injury of the blood vessels supplying the head and neck of the femur. A fracture, strain, or twist may shut off the blood supply to these vessels. It is also believed that toxic thrombosis of the vessels may play a part. Whatever the aetiology, the condition is believed to be closely related to similar changes occurring elsewhere, such as Osgood-Schlatters disease of the tibial tubercle. Scheuerman's disease in the spine, osteochondritis of tarsal and carpal bones, and other areas. By some it is believed to occur more frequently in those who have had a partial or complete dislocation of the hip joint. While it has been reported as occurring five to six times as often in males as in females, in this study it occurs equally often in both sexes. The duration is two to five years. Rest and non-weight bearing are essential to optimum recovery. Widespread necrosis of the head and neck is seen. The disintegrated bone is absorbed and replaced by granulation tissue. The head collapses and there is a characteristic deformity. There may be an outline suggestive of (1) a cap, and (2) so-called "mushroom" appearance. In well defined cases the contour of the head never returns to normal.

Symptoms

In the early stages the symptoms are evanescent and vague. The first sign noticed by parents is an inclination of the child to refrain from usual activities. He may complain of discomfort and there may be a slight limp. The child is aware of the fact that he has a hip joint where formerly he has not been conscious of the fact that one hip is different from the other. As the condition progresses the limp increases and pain becomes more noticeable. Examination shows limitation of all movements, particularly abduction and internal rotation. Flexion and adduction deformity appears. Hip and thigh muscles atrophy and the thigh appears shortened. This is seen in Figure 14. Tuberculosis, slipped capital epiphysis and congenital coxa vara give very similar symptoms. The diagnosis can only be made positively by X-ray. Even by this means, particularly in the late stage, slipped epiphysis may produce an appearance which is difficult to differentiate. Patients are rarely observed

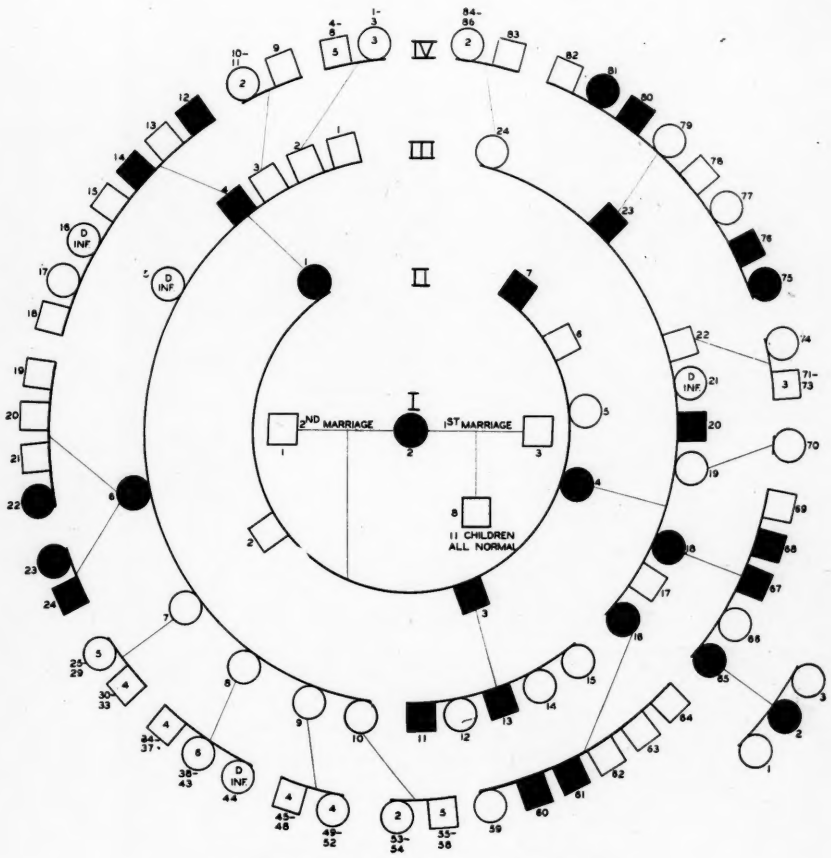
*This paper was originally planned as a joint report by the late Dr. S. C. Baldwin, Orthopedic Surgeon, and F. E. Stephens of the Zoology Department of the University of Utah. Dr. Baldwin's unfortunate death prevented his writing his part. Dr. James P. Kerby, Radiologist, has cooperated in the report in Dr. Baldwin's place. The research has been conducted under a grant from the University of Utah Research Committee.



NORMAL AND AFFECTED HIP JOINTS

Figure 10

These three X-ray photographs of the pelvic region of three affected individuals in the chart (Figure 11) show the variation in extent of the deformity of hip joints of those who had a clinical history of juvenile hip trouble. *A*—Shows the pelvis of IV-61 at 24 years of age. The appearance of the hip joints is essentially normal. Note normal contour of head, length and diameter of neck, and relation of head of femur to the acetabulum. *B*—At seven years of age both hips of IV-81 show a marked deviation from the normal. Symptoms were more severe in the left hip, though X-ray changes are more marked in the right, which shows so-called fragmentation of the head, shortening and thickening of the neck. The left hip shows a slight "thinning" of the head. *C*—At 56 years of age III-18 displays a slight shortening and thickening of the neck of the femur with a tendency to mushrooming of the head. There are slight arthritic changes of the right hip region; the left is normal.



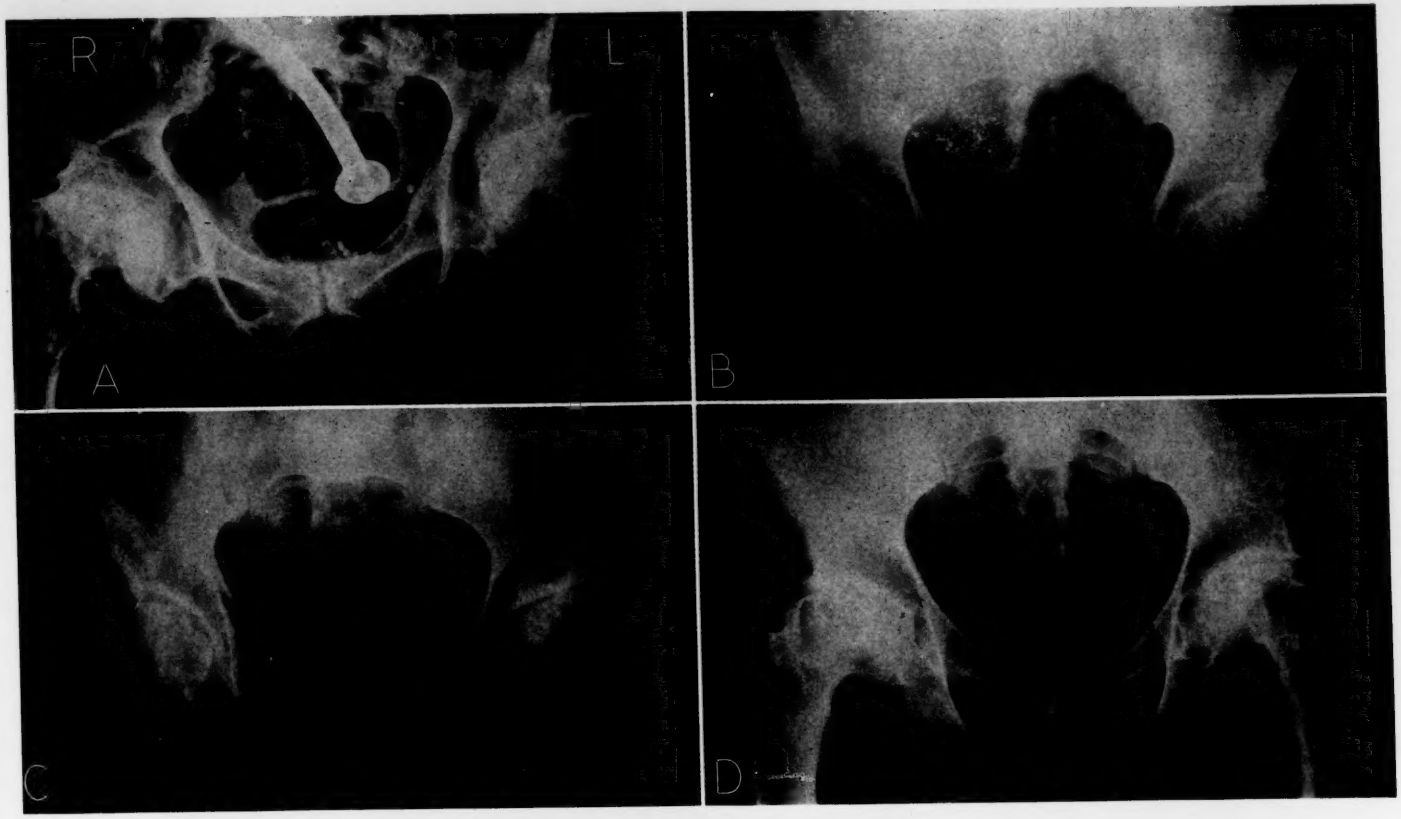
A PEDIGREE OF LEGG-PERTHES DISEASE

Figure 11

Four generations of an abnormal development of hip joints, resulting in hip dislocation and necrosis during childhood.

in the very early stage. Hence, there is no characteristic X-ray appearance although a "thinning and slight decalcification of the head arouses suspicion if the condition is kept in mind."

Typical cases show the cap or mushroom type deformity. During the course of the disease various appearances are seen. Changes in the synovial structures are not easily delineated, but some writers have mentioned prominence of the capsule at the lateral margin of the hip joint, or slight widening of the epiphyseal disc. Later, widening of the cartilaginous joint space, shallowness of the capital epiphysis, increased density of that portion of the head nearest the disc, and decalcification of the neck have been described. In well defined cases, areas of increased density appear in the head of the femur. Irregular density is seen, i.e., areas of increased and diminished density. Dense areas decalcify and undergo irregular ossification preliminary to repair. The acetabulum shows areas of increased and diminishel density and ad-



HIP JOINTS OF AFFECTED INDIVIDUALS

Figure 12

A—At 77, II-7 reveals osteoarthritic changes. Note the enlargement of the head of each femur, and the increased and diminished density in the head and neck. *B*—Individual III-20 at the age of 50 is noteworthy for the enlargement of the head of each femur, the persistence of areas of diminished density, the well-defined hypertrophic changes in the hip joints, and the tendency to extrusion of the head. *C*—The neck of the right femur of III-23 mushrooms slightly, while in the head of the left femur the mushrooming is marked, as is the thickening in the neck. Observe here the areas of diminished radiolucence. *D*—At the age of 40, IV-50 has enlargement of the head and areas of increased radiolucence in the head and neck of each femur. The proliferative change at the upper outer margin of each acetabulum is a change that is reported in middle and old age in cases having Perthes' disease in childhood.

justs its contour to conform, in part at least, to the contour of the head. The so-called fragmentation, sometimes described, is apparent, not real.

The cases reported in this paper are of special interest because in them the defect occurs as an hereditary trait which can be traced back through at least five generations. Twelve X-ray films were taken of members of the family representing four of the five generations. Typical findings are listed in the caption under each case shown.

Inheritance

There is little in the literature to suggest that Legg-Perthes' disease is hereditary. It has sometimes been observed to occur in several members of a family, but apparently no effort has been made to study the nature of its inheritance. In fact, it is not generally considered hereditary.

The present study was suggested when the trait was observed in a father and several of his children by the late Dr. S. C. Baldwin. Further investigation showed the defect in other relatives. Figure 11 gives a pedigree history of the trait. In most cases physicians made the examinations and twelve X-ray films were taken of defective individuals. In cases where it was not practicable for physicians to make the examinations, information was secured from members of the family themselves. This was carefully checked with other members of the family. It was evident that the defect is of such a nature that the family is familiar with its symptoms and are quite competent in most cases to recognize it when they see it.

The first person in this family known to have the trait was a woman (No. I-2 in the chart shown in Figure 11). She came to this country from Wales as a young married woman. She is reported by her son (II-6) and by her grand-children to have suffered from defective hips, which injury she attributed to the explosion of a boiler on board a boat on the Mississippi River. Her husband and two children were killed and she was badly injured. This occurred during her trip from Wales. She later married twice. She had one child by the second husband and seven children by the third husband. Since the trait was transmitted by this woman to four of her children, some of whom in turn transmitted it to their children, etc., it is unlikely that the explosion was the real cause of the trouble.

From the family chart it will be seen that the defect has been carried through at least five generations. No information is available regarding the parents of I-2. It is not known, therefore, whether or not the trait existed previously in the family. It has never occurred in any individual since then unless it also occurred in one of his parents.

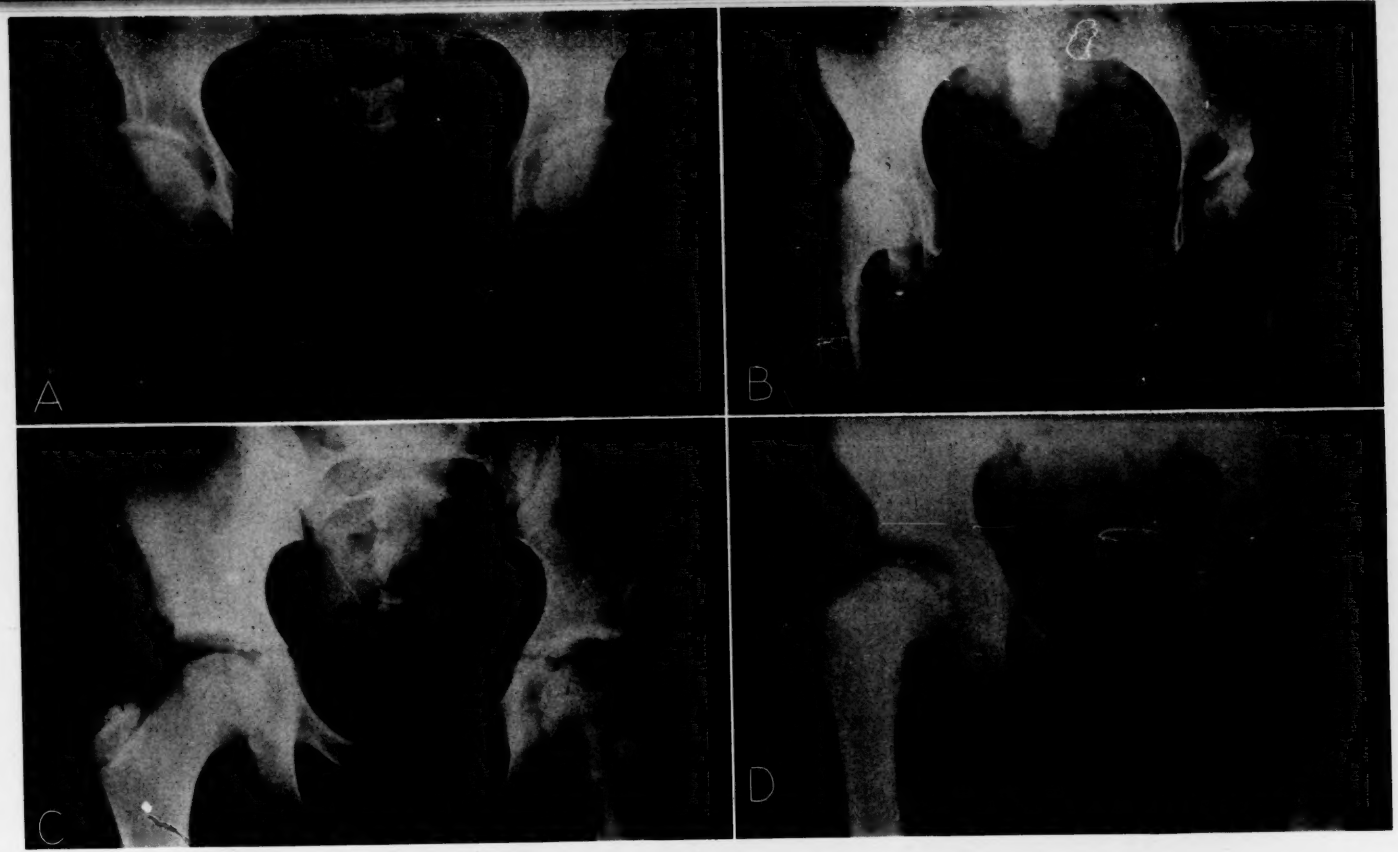
From Table I it can be seen that in the ten cases here reported, where the defect is present in one parent, 28 out of 63 children are defective. This indicated that the predisposition to develop this condition is inherited as a simple Mendelian dominant with complete penetrance. Since there is no case in which two defectives have married, all of the crosses involving this trait are simple backcrosses ($Aa \times aa$) and no opportunity is given to determine the appearance of the character in the homozygous condition. This makes it impossible to say whether or not dominance is complete.

From Table I it can be seen that the sex ratio in the families showing the trait is 35 males to 28 females. The sex ratios of defectives in these same families of 16 males to 12 females therefore indicates an equal susceptibility of the sexes for the defect. In this respect it is quite different from the cases reported by Gill who found that 85-90 percent of his cases were males.

Since Legg-Perthes' Disease is not an extremely rare condition, the question arises as to why it is hereditary in this

TABLE I.—Defective and Normal Offspring of Parents, One of Whom Has Legg-Perthes' Disease

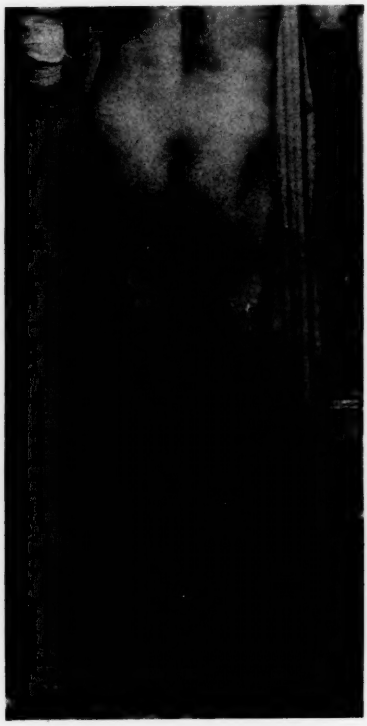
	Legg-Perthes' Disease	Normal
Males	16	19
Females	12	16
Total Observed	28	35
Calculated	31.5	31.5
Deviation	3.5±2.68	3.5



"MUSHROOMING" AND "FRAGMENTATION" OF FEMURS

Figure 13

A—At the age of 23, IV-65 shows evidence of earlier Perthes' disease. The neck of the femur is slightly thickened and shortened, while the head tends to a slight enlargement and "mushrooming." The original film shows areas of increased radiolucence in the head of the left femur. *B*—Individual IV-75 at the age of 19. There is a slight tendency to "mushrooming" of the head of each femur, and to extrusion of the femoral head from the acetabulum, as well as to a shortening and thickening of the neck of each femur. Areas of increased radiolucence are seen in the head of the left femur. *C*—Note the so-called "fragmentation" of the head of the right femur, and the shortening and thickening of the neck of the right femur. The ephiphyseal line of both femora shows slight "dentate" outline. This is IV-80, age nine. *D*—At nine years, V-4 has slight "thinning" of left femoral head, and a roughening of both acetabula.



THIGH SHORTENED

Figure 14

Photograph of III-23 reveals the effect of a more serious deformity of the left femur. See Figure 12 *C* for x-ray detail. The bone and muscle atrophy often permanently affect the bodily conformation.

particular family and yet not hereditary in the other families reported. Cases have frequently been reported since the trait was first described by Legg in 1909. In no instance, so far as the writers are aware, has it been reported as hereditary.

It seems from this that either a defective gene or some environmental condition can be responsible for Legg-Perthes' Disease. In many instances, undoubtedly, no attempt has been made to obtain a family history of the cases reported and therefore the hereditary basis has not been recognized. In others the real cause may have been masked by an improper diagnosis. Perhaps a different type of inheritance is responsible for the defect in some families so that only rarely does the trait express itself. Several defects and deviations are known which, though phenotypically undistinguishable are conditioned by different genes. Thus dominant and recessive polydactyly are recognized. Possibly a recessive gene, rather more frequent in its distribution, causes some of the sporadic cases.

It is quite unlikely that each new case is due to a separate mutation. If this were true, it would be transmitted to the offspring and its hereditary basis would be apparent since Legg-Perthes' Disease does not prevent reproduction. In the light of all this, it would seem that the character may be either hereditary or environmental in nature. Undoubtedly, in the family reported in this paper, the condition originated at some time as a dominant mutation which has been passed on ever since in a simple Mendelian manner.

There is considerable variability in the expression of the defect. In some cases it is slight while in others it is very severe, leaving the affected individual a permanent cripple. No. III-23 says it is not unusual for him to "throw his hip out of place" as the result of some simple act such as tying his shoes. This variability in expressivity may be due to genetic differences or may depend largely upon the degree of activity of the child while the bone is soft and capable of being deformed by bearing the weight of the child's body.

As previously stated, so far as our record of this family reveals, no marriage has occurred between two individuals showing Legg-Perthes' Disease. Each defective individual, therefore, would be heterozigous for the defect since one parent is always normal. Each child born to a defective parent would have an equal chance of inheriting the trait. Normal children of defective parents could not transmit the trait since they have not received the defective gene. If the defective individuals would fail to

The epiphyseal line of both femora shows slight dentate outline. This is IV-80, age nine. *D*—At nine years, V-4 has slight "thinning" of left femoral head, and a roughening of both acetabula.

reproduce, the defect could be eliminated from this family in one generation.

Summary

In many cases Legg-Perthes' Disease occurs without any family history showing the defect. This suggests the possibility that these cases are non-hereditary or are due to some type of heredity which produces the defect at less frequent intervals.

In this paper, cases are reported showing Legg-Perthes' Disease transmitted through five generations. It is inherited as a simple dominant trait having complete penetrance. The expressivity of the trait is variable.

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